IN THE UNITED STATES DISTRICT COURT
FOR THE NORTHERN DISTRICT OF MISSISSIPPI
GREENVILLE DIVISION

OSCAR BROOKS	**PLAINTIFF**
V.	**CASE NO. 4:07CV62**
INGRAM BARGE COMPANY **JANTRAN, INC.**	**DEFENDANTS**

MEMORANDUM OPINION

This cause comes before the court on the motions in limine [51, 59] of the defendants, Ingram Barge Company ("Ingram") and Jantran, Inc. ("Jantran") to exclude the scientific expert testimony offered by the plaintiff, Oscar Brooks.

Facts

Brooks worked for Ingram and Jantran aboard vessels operating along the Mississippi River and interlocking waterways for a period of twenty eight years. During those years of service, Brooks was exposed to diesel exhaust while onboard the vessels. He also smoked three packs of cigarettes a day for approximately fifty years. In 2004, Brooks was diagnosed with lung cancer. He then filed suit against his former employers alleging that exposure to diesel exhaust played at least some role in his developing cancer.

In seeking to prove his claim, Brooks relies on the testimony of two expert witnesses, David F. Goldsmith, MSPH, PhD and Louis Hamer, M.D. The question before the court is whether these experts' opinions are reliable and relevant as defined by Federal Rule of Evidence 702. In trying to resolve that question, the court ordered a *Daubert* hearing.

Standard of Review

The Supreme Court has charged trial court judges with the responsibility of acting as

gatekeepers in determining when expert testimony is admissible. *Daubert v. Merrell Pharmaceuticals, Inc.*, 509 U.S. 579, 589 (1993). *Daubert* requires that "when expert testimony is offered, the trial judge must perform a screening function to ensure that the expert's opinion is reliable and relevant to the facts at issue in the case." *Watkins v. Telesmith, Inc.*, 121 F.3d 984, 988-89 (5th Cir. 1997). Determining reliability requires assessing "whether the reasoning or methodology underlying the testimony is scientifically valid." *Daubert*, 509 U.S. at 592-93. Relevance rests on "whether [that] reasoning or methodology properly can be applied to the facts in issue." *Id*. at 593.

Daubert set forth a non-exclusive checklist for trial courts to use in determining the reliability of expert testimony. *Daubert*, 509 U.S. at 593. Those factors include: (1) whether the expert's technique or theory has been tested; (2) whether the technique or theory has been subjected to peer-review and publication; (3) the known or potential rate of error applicable to the technique or theory; (4) the existence of standards and controls applicable to the technique or theory; and, (5) whether the technique or theory is generally accepted in the scientific community. *Id*. at 593-95. In performing this analysis, a court must determine an expert "employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field" before finding the evidence admissible. *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 152 (1999).

Analysis

The arguments presented to the court raise issues of causation. In proving his case, Brooks must show both general and specific causation. *Knight v. Kirby Inland Marine, Inc.*, 482 F.3d 347, 351 (5th Cir. 2007). General causation here would require proof diesel exhaust is

capable of causing lung cancer. *See id*. (citing *Merrell Dow Pharm., Inc. v. Havner*, 953 S.W.2d 706, 714 (Tex. 1997)). Specific causation requires proof diesel exhaust caused Brooks' lung cancer. *See id.*

Dr. David F. Goldsmith

The well qualified Dr. Goldsmith is an associate research professor at George Washington University. His specialty is in occupational and environmental epidemiology and public health specifically as it relates to lung cancer and other pulmonary health effects. The majority of Goldsmith's work in this field deals with exposure to silica.

Goldsmith came to his conclusions working within the field of epidemiology. He defines epidemiology as the "basic science of public health." Epidemiology looks at causes of disease in groups of people with a focus on how risk factors relate to disease. No one doubts Goldsmith is qualified to evaluate epidemiological studies and draw conclusions from those studies.

In the instant matter, Goldsmith reviewed Brooks' self-reported work history and the epidemiological evidence available. From this information he made three conclusions: (1) diesel exhaust can cause lung cancer; (2) diesel exhaust works synergistically with cigarette smoke to cause lung cancer; and, (3) diesel exhaust played a role in causing Brooks' lung cancer. The first two conclusions relate to general causation. The third conclusion is related to specific causation.

General Causation

Goldsmith's conclusion that diesel exhaust can cause lung cancer is based on a number of studies including most importantly studies showing links between exposure to diesel exhaust in railroad workers and long-haul truck drivers and the development of lung cancer in those groups. These studies, while not definitive, show a correlation between occupational exposure to diesel

exhaust and incidence of lung cancer.

This is supported by epidemiological research showing a relative risk of between 1.3 and 1.7 for exposure to diesel exhaust and the contraction of lung cancer. These numbers are based on a scale with three possible outcomes. A substance can either be classified as having a link of less than one, meaning the substance is actually a cancer preventative such as an antioxidant. A substance can have a link of one, meaning there is no increase or decrease in cancer occurrence with exposure. Finally, a substance can have a value of greater than one, meaning that exposure increases the likelihood of developing cancer. Goldsmith classified diesel exhaust exposure as a moderate elevated risk stating that a high risk of developing cancer would require a relative risk greater than 2.0.

Goldsmith's conclusions about relative risk are backed by a number of consistent studies. For example Diesel Exhaust Exposure and Mortality Among Males in the American Cancer Society prospective study published by Paolo Boffetta, MD, Steven D. Stellman, PhD, and Lawrence Garfinkel, MA in Volume 14 Issue 4 of the American Journal of Industrial Medicine showed a relative risk of 1.24 for truck drivers,[1] 1.59 for railroad workers, and 2.60 for heavy equipment workers. A Case-Control Study of Lung Cancer and Diesel Exhaust Exposure in Railroad Workers published in a 1987 volume of The American Review of Respiratory Disease showed a relative risk of 1.41 for railroad workers under the age of 64 with ten years railroad

[1] Overall truck drivers exposed to diesel exhaust did not have a higher cancer rate than those not exposed. However, the researchers noted a trend showing risk for truck drivers did increase with the increased exposure to diesel exhaust.

experience.[2]

Additionally, in June of 1998 the Air Resouces Board and the Office of Environmental Health Hazard Assessment for the state of California prepared an initial statement of reasons for rulemaking proposing to identify diesel exhaust as a toxic air contaminant. In making this report the two agencies reviewed more than thirty epidemiological studies finding they provided evidence "that long-term occupational exposures to diesel exhaust were associated with a 40 percent increase in the relative risk of lung cancer." They analyzed the findings of the epidemiological studies and concluded these findings were not likely due to bias or chance.

The International Agency for Research on Cancer ("IARC") classifies substances in four categories. Group one is for substances known to be carcinogenic to humans. Group two is divided into two subcategories. Group two(a) is for substances in which there is strong evidence of probable carcinogenic effects on humans. Group two(b) is also for substances that are probably carcinogenic to humans, but where the evidence is weaker. Group three substances are those where the evidence is too limited or inadequate to make a reasoned decision about its carcinogenic effect. Group four substances are probably not carcinogenic to humans. The IARC 1998 monograph on the evaluation of carcinogenic risks to humans classifies diesel exhaust in group two(a).

Goldsmith also relied on animal studies showing there is a dose response to diesel exhaust exposure and the development of lung cancer.[3] According to Goldsmith's testimony

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[2] This study did not show an increased risk for workers over the age of 64. The researchers concluded this was because many of the workers in that category retired shortly after the railroads switched to diesel engines.

[3] The California report on proposed identification of diesel exhaust as a toxic air contaminant also has an analysis of available animal studies. However, the report states the agencies involved "concluded that available

these studies conclude the more exposure to diesel exhaust, the greater incidence of lung cancer. However, Goldsmith stated that it was impossible to know what the dose response in human exposure was. This difficulty in finding a dose response relationship is supported by the report of the Air Resources Board and the Office of Environmental Health Hazard Assessment which states they "recognize that the limited exposure information available contributes to the uncertainty of the dose response risk assessment based on the human studies."

Goldsmith did testify that in his opinion exposure as a tug boat worker of only five to seven years would not meet the temporal cogency point. Based on this testimony, Goldsmith's opinion is that for diesel exhaust to cause cancer there must be an extended period of exposure. This is consistent with the findings of a number of studies and articles. For example the article Diesel Exhaust published by Howard Frumkin, MD, Dr.PH and Michael J. Thun, MD in the Cancer Journal for Clinicians urges patients be informed:

> [i]f you are or have been heavily exposed to diesel fumes your risk of lung cancer may be increased. If you have been exposed to diesel fumes in the general environment, the increase in your risk is likely to be very small. If you have had prolonged exposure to high concentrations at work, your risk is higher.

These studies support the conclusion that diesel exhaust can cause cancer. There is, however, conflicting evidence in the literature. The court does not know, nor should it determine if diesel exhaust causes cancer. However, the totality of the available information is enough under the *Daubert* standard to allow an expert who has parsed that information to testify that diesel exhaust can cause lung cancer. Goldsmith's theory has been tested, it has been subjected to peer-review and publication, and standards and controls were in place for the best studies

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human data lend[s] more confidence in the prediction of human risks than the data from the available animal studies because of the uncertainties in the animal studies and of extrapolating [risk] from animals to humans."

available. While experts themselves may disagree about his conclusion, both sides of the disagreement can reasonably support their opinions with reliable scientific data. Additionally, it appears from the literature that a majority of the scientific community believes that diesel exhaust can cause lung cancer.

Goldsmith next puts forth the proposition that diesel exhaust and cigarette smoking work synergistically to cause lung cancer.

This conclusion appears to be drawn exclusively from one study on Swedish truck drivers.[4] That study, Professional Driving, Smoking, and Lung Cancer: A Case Referent Study, was originally published in Volume 42 of the British Journal of Industrial Medicine. The study states "[n]umerically, a synergism was found between occupational exposure [to diesel exhaust] and smoking." However, the authors of the study point out the small pool of information available from which to make this conclusion.

The researchers looked at 467 men under the age of eighty who drove trucks and died of lung cancer compared with 467 men of the same age and from the same towns that died of other causes excluding suicide and lung cancer and 467 men of the same age and from the same towns who were still living. The researchers themselves seem unsure what exactly their results meant. They write:

> Smoking drivers in the group aged 70 and over seemed to have a considerably increased risk compared with smoking non-drivers in the same age group. Numerically, a synergism was found between occupational exposure and smoking with a synergism index of 2.42 if the material with two referents was used and of 1.73 if the whole material with one referent was used. The estimated relative risks obtained when the whole material was analyzed . . . were lower than when only

[4] Footnote five of this study points to another study which may support this conclusion. Goldsmith, however, makes no mention of this other study.

> the cases with two referents were used . . .. the reason is probably the different composition of the referent groups. The studied exposures (smoking and occupation) may cause an additional risk concerning causes of deaths other than lung cancer. Comparison with deceased referents may therefore underestimate the lung cancer risk. The opposite may occur when living referents are used as these may represent a positively selected group concerning disease risk. The analyses with two referents (one deceased and one living) might therefore have given the best estimates.

This statement indicates that the study has shown a numerical correlation from which more research is needed. The relative risk swing between the comparison models is quite expansive leaving open the question of which model is more correct. Additionally, the researchers point to problems with both models in developing a definitive answer. These problems indicate that further studies and theories are needed to develop reliable science. Goldsmith fails furnish this court any additional research contemplating the synergy between diesel exhaust and cigarette smoking.

Based solely on this data, the court can not accept this conclusion as meeting the requirements of *Daubert*. This court is not prepared to begin making scientific determinations. However, it is clear that any evidence tending to show a synergy between diesel exhaust and cigarette smoking is very limited. The theory itself has been tested and published in a peer reviewed journal. The researchers though admit there are problems with the controls in the study. The number and type of individuals in the study are minute. Additionally, nothing else this court has seen in the literature stands for this proposition. The court is not convinced that an eminent epidemiologist such as Goldsmith would rely on this limited data to make a broad conclusion in his normal professional life. This conclusion is not based on a rigorous intellectual process, but is instead speculative. As such it is inadmissible under Federal Rule of Evidence

702.

Specific Causation

The question now before the court is whether Goldsmith can testify to a degree of scientific certainty that diesel exhaust caused Brooks' cancer.

Goldsmith is not a specialist in the effects of diesel exhaust. He admits that he has basically no knowledge of how commercial boats operate. Even with these two handicaps he has no trouble testifying that diesel exhaust caused Brooks' cancer.

However, Goldsmith's testimony is not well supported. Goldsmith's initial opinion, which seems to have changed little, was made with no knowledge that there were any measurements of diesel exhaust exposure as it relates to commercial boats. After being informed of a study measuring such exposure, Goldsmith's testimony did not change. However, that study by Dr. Morton Corn showed the levels of exposure to be safe.

Goldsmith reveals he is unsure of any numerical amounts of exposure Brooks received.[5] As discussed above he was also unable to point to a dose-response level when asked for this information by the court. The court asked Goldsmith at what level of exposure he could say to a reasonable scientific certainty that diesel exhaust caused someone's cancer. His answer is telling. Goldsmith responded in part:

> To quantify the actual amount, we have to be cognizant of the fact that there's really two things that are going on. One is what the person's genetic background

[5] Goldsmith did eventually come to estimate a level of exposure. That estimate was stricken by the court in a separate order due to its untimeliness. In deciding to strike Goldsmith's additional testimony, the court was mindful that Goldsmith seemed to be working the scientific method backwards. That is, he came to the conclusion he wished to state in this court and then kept adding new information in an attempt to support that conclusion to the point this court would accept it. The court finds this is not the same level of intellectual inquiry Goldsmith would use in developing studies or opinions in his field. This method offers no assurance of reliability, but instead allows an expert to mold his supporting evidence in order to testify to any conclusion he wishes.

> is and secondly what his exposure is in the workplace. . . . [W] haven't got specific information about specific individuals and we can't follow them up to the degree that we would like to be able to do. I can, if I had a lot more time, calculate what I assume to be the safe level of exposure to diesel exhaust assuming some degree of exposure on boats, on tug boats, compared to the diesel exposure in the Garshick studies and then we can see how far away or how close that is to what Mr. Brooks lifetime exposure, thirty-three years or twenty-eight years was assuming some degree of average amount of diesel exposure given his job.

This answer shows that Goldsmith simply does not know everything he needs to in order to determine if Brooks' cancer was caused by diesel exhaust. Goldsmith acknowledges that it is unclear how much exhaust Brooks was exposed to, how much exhaust it takes to make developing cancer a probability, or how much other factors played a role in Brooks developing cancer.

It is clear that other factors likely did play a role in Brooks' cancer developing. Brooks was a heavy smoker for fifty years. Everyone involved in the case agrees that smoking cigarettes is likely to lead to lung cancer. The article, Epidemiology of Lung Cancer, submitted by the defendants states "in the United States, active smoking is responsible for 90% of lung cancer cases." Additionally, that article found "[t]he risk of lung cancer among cigarette smokers increases with the duration of smoking and the number of cigarettes smoked per day." These finding are consistent with other well supported literature on smoking and lung cancer. As a heavy smoker Brooks was at least twenty times more likely to develop lung cancer than a non-smoker.

While Goldsmith stated that he took this risk factor into consideration, he appears to simply explain it away with the conclusion that smoking and diesel exhaust work synergistically to cause lung cancer. The court has already rejected this conclusion. As such, Goldsmith's

accounting for smoking as a risk factor is insufficient to pass even the gatekeeping function of *Daubert*. Further, even if the court were to accept that cigarette smoking and exposure to diesel exhaust synergistically caused cancer, there is not enough information to establish this synergistic effect caused Brooks' lung cancer. The minimal research in this area does not illuminate a point at which the two substances together cause cancer. Under this analysis it is still unclear what Brooks' exposure levels were. Absent this information one cannot determine if this effect contributed to Brooks' cancer. No reliable science has been presented to this court to support an opinion that an individual in the highest risk category was exposed to some unknown amount of a moderate carcinogen and to then find that the moderate carcinogen caused his cancer is simply unreliable.

Finally, Goldsmith's testimony is based on further speculation. He makes clear that genetics play a role in the contraction of lung cancer. In fact at least some cases can be found in which individuals who do not have known risk factors develop lung cancer. However, Goldsmith admits he has no knowledge of Brooks' genetic makeup or predisposition to contract lung cancer. Instead Goldsmith simply assumes that Brooks' genetic makeup is average.

As it relates to specific causation, Goldsmith's opinion is based too heavily on speculation and guesswork. There is not enough scientific evidence to bridge the analytical gap between the fact that diesel exhaust can cause lung cancer and Goldsmith's subjective belief that diesel exhaust caused Brooks' cancer. As such, no specific causation evidence exists. This renders Goldsmith's opinion unreliable and inadmissible under Federal Rule of Evidence 702.

Dr. Louis Hamer

Dr. Hamer is a pulmonogist with excellent credentials. However, his testimony reveals

that his practice is in the diagnosis and treatment of disease as opposed to the causes of disease. This shows in the way that Hamer came to his conclusion. He conducted significantly less research in making his conclusion that diesel exhaust causes cancer than did Goldsmith. However, because the court has already decided to accept Goldsmith's testimony on general causation, it will accept for the purposes of this motion the fact of general causation as true and established. Thus the court will only examine Hamer's conclusions as they relate to specific causation; that is whether diesel exhaust caused Brooks' cancer.

Specific Causation

The court finds little need to examine Hamer's method. Hamer's testimony clearly shows that his conclusion is not reliable to any scientific certainty. Time and time again, Hamer insists that his belief is subjective, the data he used to come to that belief is subjective, and that there is no way to accurately draw a line between individuals who were exposed to diesel exhaust and did not contract lung cancer and those that did. Hamer considered that Brooks had been exposed to a large amount of diesel exhaust. Hamer's own testimony, however, makes clear that he is uncertain of the amount of exposure. He states:

> large is a subjective term and the history of exposure in itself is subjective. Anytime we're taking a history of exposure to any toxin, whether it's diesel exhaust, asbestos, silica, you know, cigarette smoke, there's always going to be a subjective component. . . . Because large is a subjective term. To me, twenty-eight years working on tug boats is a large number. Twenty-eight years sounds like a long time to me. . . . I think that the degree of exposure is a subjective analysis. As much as we'd like it to be scientific, as much as we'd like it to be mathematical, in the end it is subjective.

Hamer's testimony that his exposure reliance is uncertain and not scientific dooms his conclusion.

As a gatekeeper, this court must keep out speculative and unfounded conclusions such as the one offered by Hamer. To allow a subjective opinion to be submitted to a jury as an objective finding would violate both the letter and spirit of *Daubert*. Hamer's testimony is inadmissible.

Conclusion

The experts offered by Brooks are simply unable to make the leap from diesel exhaust causes cancer to diesel exhaust caused Brooks' cancer. This failure may be in the scientific data available or in the application of that data to the facts at hand. Nevertheless, without more information they can not bridge the gap between general and specific causation. Their testimony is thus inadmissible. The motions of the defendants to exclude the plaintiff's experts are GRANTED.

This the 21st day of November, 2008.

/s/ MICHAEL P. MILLS
CHIEF JUDGE
UNITED STATES DISTRICT COURT
NORTHERN DISTRICT OF MISSISSIPPI